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APPARATUS AND METHOD FOR CHANGING CRITICAL BRAIN ACTIVITY USING LIGHT AND SOUND

TECHNICAL FIELD OF THE INVENTION

The present invention relates to the field of critical brain stimulation, and more particularly, to an apparatus and method for the treatment of neural diseases through the controlled, targeted use of light and/or sound depending on the underlying physiologic pathology of a patient.

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This application claims priority as a C-I-P of United States Patent Application Serial No. 09/201,093, filed November 30, 1998, US Pat No. 6,299,632.

RL 4/5/12

BACKGROUND OF THE INVENTION

Without limiting the scope of the invention, its background is described in connection with the use of light therapy to stimulate cerebral plasticity across hemispheres, as an example.

Light has been shown to effect the stability of a person's energy, mood, sleep, concentration and the regulation of a person's circadian rhythms. Light deprivation, for example, has been shown to cause fatigue, irritability, anxiety, weight gain, social withdrawal and a lack of alertness.

The human brain produces detectable signals that vary in strength and frequency over time. These signals are detectable as electromagnetic waves, and vary from one part of the brain to another and may, in fact, vary over time. Electromagnetic waves with different frequencies are associated with different moods and mental abilities.

It is generally believed that a person afflicted with a sleeping disorder has problems generating a delta rhythm. In contrast, people who have difficulties learning or display behavioral problems that affect learning, have problems

associated with abnormalities of the alpha rhythm. These rhythms have been found to be regulated by brain biochemistry.

The use of light therapy, unlike drug therapy, has been used to treat patients that have been afflicted with a seasonal affective disorder. United States Letters Patent No. 5,562,719 to Lopez-Claros describes one such method for treating seasonal affective disorders that relies on preferential light stimulation in a hemifield pattern only affecting one quadrant or 50% of the non-dominant cerebral hemisphere. Seasonal affective disorder is a condition that affects from 5-20 % of the population in areas with decreased year time light levels, such as the Northern Hemisphere. A common treatment for seasonal affective disorders is the use of lamps or light boxes that provide between 2,500 to 10,000 lux illumination in a hemifield pattern only affecting the inferior quadrant or 50% of the non-dominant cerebral hemisphere. The use of level illumination is an attempt to stimulate summer-like light levels. The Lopez-Claros patent is directed to the stimulation of one cerebral hemisphere to a greater degree than the other, thereby treating seasonal affective disorder by preferential light treatment. The Lopez-Claros invention, however, stimulates only one quadrant of the optical axis or 50% in both eyes thereby precluding its use in the stimulation of a hemisphere wholely.

As apparatus and method for treating an individual by electroencephalographic disentrainment feedback is the focus of United States Letters Patent No. 5,365,939 issued to Ochs. Electroencephalographic disentrainment feedback involves measuring a patient's brain waves, and based on those brain waves, generating impulses that disrupt brain waves by "disentraining" brain waves that are "entrained" or entrenched in the brain. By "disentraining" the entrenched brain waves, a patient's sub-optimal posttraumatic neural functioning is restored. The apparatus, however, requires the supervision of a doctor, as people with hypersensitivity may require that treatment be immediately stopped. The increased supervision makes the cost of use very great and eliminates its portability.

A slight variation from the Ochs patent, is United States Letters Patent No. 5,036,858 issued to Carter and Russell, that involves using two frequencies, with a slight frequency differential between the two frequencies, to change a patient's brain waves. The patient's brain waves are also measured, and based on these measurements, disruptive brain impulses are generated that disrupt the patient's brain waves in a constant feedback mechanism.

SUMMARY OF THE INVENTION

The invention disclosed herein is an apparatus for selectively stimulating target regions of the cerebral hemisphere using a controlled light and sound generating apparatus. The present invention helps address the present need for an apparatus and method that enhances the innate abilities of individuals with limbic disorders.

Also needed is an apparatus and method that is simple to use, inexpensive and portable, thereby providing greater distribution to those most in need of treatment. One example of individuals who may benefit the most from an inexpensive, simple to use device that increases learning abilities are patients and individuals in poor, urban, inner cities and in rural areas. More specifically, what is needed is a simple, inexpensive device that may be used by, e.g., elementary and secondary school children whose parent, or guardian, can not afford expensive drug treatments requiring medical supervision. Others that may benefit from such an apparatus and method are athletes, business people, and academicians.

In one embodiment the apparatus of the present invention is a device that covers the patient's eyes, such as a pair of sunglasses. Other devices may use similar technology in order to enhance ones ability to mentally focus. These devices include sports helmets that are used to protect players'

craniums and may be integrated into the protective head gear, e.g., football, hockey, baseball, racing car, motorcycle and bicycle helmets.

Another application of the technology is with computer monitors and televisions. This embodiment encompasses one or more oscillating lights set-up in a proscribed manner on a person's computer monitor. The light pattern of the present invention may be displayed in a subliminal alternating checkerboard pattern that would be set to the individual user. In one embodiment, the light will stimulate the non-dominant cerebral hemisphere greater than the dominant cerebral hemisphere. In this version, the non-dominant cerebral hemisphere is stimulated to a greater degree than the dominant cerebral hemisphere. It is the coordinated stimulation of the non-dominant hemisphere that helps create a balance of integration of excitatory post synaptic potentials (EPSP).

The apparatus for selectively stimulating the non-dominant cerebral includes a surface placed in close proximity to a patient's eyes and one or more lights disposed on the surface. The one or more lights stimulate the eye connected to the non-dominant cerebral hemisphere to a greater extent than the eye connected to the dominant cerebral hemisphere at a rate of approximately, e.g., 60/40. By overstimulating the non-dominant hemisphere there is an increase in the patient's

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ability to maintain a heighten mental status, and in turn sets up for a globality of increased muscular activity.

Alternatively, the surface may be sleeping goggles. In yet another embodiment of the present invention, the glasses reflect light from a source next to the eye (light is reflected from the glass surface) into the patient's eyes.

The types of light that are used with the apparatus and method of the present invention may include white light, plane polarized light, or light that varies in color. The timing and intensity of the light may be controlled by a microcontroller or by an operator.

BRIEF DESCRIPTION OF THE DRAWINGS

For a more complete understanding of the features and advantages of the present invention, reference is now made to the detailed description of the invention along with the accompanying drawing in which corresponding numerals in the different figures refer to corresponding parts and, in which:

Figure 1 is a presentation of the neural anatomy of the optical system and the relation of the visual fields;

Figure 2 is a schematic representation of the optical stimulant of the present invention; and

Figure 3 is a schematic representation of the optical stimulant in a hemifield pattern;

Figure 4 is a writing sample from the patient in case study #1 before exposure to the present invention;

Figure 5 is an 8-bit bidirectional I/O port microcontroller that provides current to the light sources;

Figure 6 depicts a rear elevation view of a device employing the features of current system; and

Figure 7 is a flow diagram of the process steps that may be executed by the code embedded in microcontroller in accordance with one embodiment of the present invention.

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DETAILED DESCRIPTION OF THE INVENTION

While the making and using of various embodiments of the present invention are discussed in detail below, it should be appreciated that the present invention provides many applicable inventive concepts which can be embodied in a wide variety of specific contexts. The specific embodiments discussed herein are merely illustrative of specific ways to make and use the invention and do not delimit the scope of the invention. The present invention has been developed based on empirical data obtained from patients. The underlying physiologic basis of the results, as disclosed hereinbelow, in now way should limit the scope of the invention.

The structures involved in the limbic system are the ventral mesencephalic tegmental mesolimbic neuronal pools called A10, which project into the amygdala, ventral putamen, ventral caudate, cingulate and the hippocampus. Presently, the only physical-clinical way to understand the integrity of a person's limbic reality is to conduct an accurate clinical history and to assess the function of the A10 cells. When A10 cells fire they produce dopamine, which is important of the survivability of the ventral neostriatum.

When mesencephalic A10 cells fire, they stimulate ventral neostriatal inhibition of pallidal inhibition of the motor thalamus. The stimulation of the ventral neostriatum allows

the motor cells of the thalamus to reach threshold and fire the somesthetic strips 3,1,2, of the post-central gyrus, which in turn drive the primary, supplementary, and premotor strips 3,1,2, of the post-central gyrus. The stimulation of premotor strips 3,1,2, of the post-central gyrus, in turn, drive the primary, supplementary, and premotor strips of the precentral gyrus all 4, 4s, and 6. The consequence of this chain of neuro-stimulatory events is that, as a result of motor activity, these motor cells act in concert with emotional events.

When something in the environment evokes an emotional response, cells in the cerebellum fire before cells in the cortex. When cells fire in the cerebellum, these cells in turn fire monosynaptically to the A10 cells and to the pontine epinergic cells (Locus cerulcus A2, A4, and the sub cerulcus A1, A3, A5, A7). It is the motor event, therefore, that causes an emotional event to fire cells at the A10 loci.

It is based on this series of observations, in conjunction with the results demonstrated herein using the present invention, that it is now recognized that the controlled activation of the A10 cells allows a person to smile at something cute and cuddly, to laugh outwardly at a joke, etc. There are large postsynaptic pools from the A10 cells within the amygdala and the anterior cingulum. When

cells in the amygdala and anterior cingulum are appropriately stimulated, they cause cells in the ventral neostriatum to fire, which in turn inhibit the release of the substantia nigra pars reticulatas and the globus pallidus pars intemus. The release of inhibition by the substantia nigra, pars reticulatas and the globus pallidus pars intemus releases the motor thalamus from inhibition resulting in a motor event.

Therefore, an overactive mesolimbic system may produce non-ballistic motoric activities without blepharospasm and with or without pupillary plasticity. It has been observed that an overactive mesolimbic system is a consequence of amygdala and cingulum activation of the neostriatum. Individuals with an overactive mesolimbic system are very spontaneous in regards to their motoric control. These patients tend to be very sarcastic and speak without thinking and potentially regret what they just said. For example, they may laugh at a funeral. These patients are also within the segment of the population that experience road rage with the concomitant motoric expression visualized by adjacent motorists as "flipping the bird" or screaming obscenities. A sub-segment of these patients also tend to present with angulations and dystonias of the left upper extremity due to aberrancies in right limbic expression with a probability of wind-down with specific basal ganglionic degeneration in the

right cerebral cortex. It should be noted, however, that not all overactive mesolimbic patients experience any of these motoric overindulgences.

An underactive mesolimbic system may result in decreased amygdalal activation, which may decrease the neostriatal inhibition of the substantia nigra pars reticulatas and the globus pallidus pars intemus resulting in a shift towards K+ equilibrium potential of the motor neurons of the thalamus. The result may be a loss of facial expression to the left side of the face.

A major problem in schools throughout the country is poor performance and discipline problems, caused in part by the learning disabilities of students. It has been estimated that at least 40 million Americans exhibit some symptoms of dyslexia, including up to 20% of all students. The results obtained using the non-invasive, non-pharmaceutical therapeutic treatment disclosed herein provide a radical new alternative for treating learning disabilities and other limbic disorders.

Learning begins at birth and involves the development of the brain hemispheres driven by the ability to perceive one's environment through the sensory system, i.e., touch, sound, smell, taste and vision. Children will thrive when they receive the proper sensory inputs and when signal pathways in

the brain develop correctly. A lack of proper sensory inputs or incomplete development results in poor cognitive capability that manifests itself in poor learning skills. It is a major cause of ADD, ADHD and dyslexia.

The apparatus and method disclosed herein, named, EYELIGHTS, may be used for reconfiguring or redirecting nervous impulses to treat the symptoms of dyslexia, Attention Deficit Disorder (ADD) and Attention Deficit Hyperactive Disorder (ADHD). The symptoms of these diseases may be minimized or eliminated in many students, as demonstrated hereinbelow, and possibly in the general population. All the studies disclosed herein were conducted in a doctor-patient relationship under controlled conditions after having obtained full consent from the participants.

The problem of learning disabilities is a plague on US schools. For example, the "Attention Deficit Disorder" page in the "www.ONHEALTH.com" web site, assessable through "www.SNAP.com", states that as many as 20% of all school children are affected with ADD and ADHD. While many more boys are diagnosed with the problem, they state that it is becoming clear that ADD also affects many girls. Their difficulty lies mostly in inattention without hyperactivity as with boys and so the diagnosis may be overlooked. It is generally known in

the art that common symptoms in these disease conditions include:

1) Habitual failure to pay attention;

2) Difficulties with school work;

3) Excessive distractability;

4) An inability to organize, even with activities that are enjoyed;

5) Difficulty following instructions and repeated failure to complete projects;

6) Impulsiveness;

7) Hyperactivity - fidgeting and running about; and

8) Excessive talking and frequent interrupting.

Dr. Harold Levinson, a widely published and reknowned expert on dyslexia, hosts a world wide web page at "dyslexiaonline.com". At this site Dr. Levinson states that over 40 million children and adults have symptoms of dyslexia. One of the most striking aspects of Dr. Levinson's research is the fact that over 78,000 visits have been made to the site since September 1998, approximately 11,000 visits per month, looking for help or information.

A very important feature of the present invention has been the ability to demonstrate an improvement in the cognitive capability of a participant, e.g., students, through the use of light stimulation to the non-dominant hemisphere of

the participant's brain. The results demonstrate that the apparatus and method of the invention provide a unique therapeutic benefit to the participants as shown by an improvement of cognitive skills.

The results disclosed herein also demonstrate a reduction or almost complete elimination of the symptoms of ADD, ADHD and dyslexia in participants at the elementary school level. The method also provides a means of determining the mechanism for the effect that heretofore would have required extensive invasive procedures on test subjects. The physiologic mechanism uncovered using the present invention involves the relationship that exists between physiological blind spot mapping (PBSI) and Caldwell's Visual Evoked Potentials (VEP). The invention was used to demonstrate the effectiveness of VEP in the diagnosis and treatment of ADD, ADHD and dyslexia.

Building on the knowledge base as the foundation for commercialization of the therapy and supporting hardware, the apparatus and methods disclosed herein may be used in a wide variety of applications. Such treatments include commercial applications through learning centers, optometrists, neurologist, kinesiologists, psychologist and others with the scientific and medical background to perform the diagnostic testing and manage the necessary treatment.

Hemispheric dominance is potentially a source of many learning problems. Hemispheric dominance is driven by the development of the visual system. Vision is one of the first systems to develop in young child, therefore, the visual system is the most important input systems in the development of the neuraxis. The development of an infant's eyes, e.g., is an indication of the impact of integration of midline brainstem nuclei upon the muscle development or control systems of the eyes.

As the development of the visual axis occurs, a corresponding development of the erector spinae musculature is observed during development. In this event, the child begins holding its head up, and progresses to sitting up, crawling, and finally standing up prior to walking. The visual system input toward the development of physical strength and muscle control are based primarily on thalamocorticocerebellar feedback loops. Therefore, visual stimulation and the concomitant development of the thalamocorticocerebellar brain centers may dictate the development of the brain. As the nervous system develops after birth, a noticeable disparity begins to develop in the midline structures of the child's visual system. Once midline development occurs, one of the hemispheres of the brain begins to become dominant.

The development of the midline structures occurs such that the brainstem nuclei that first develop will be those that are most medial, viz., cranial nerves 3, 4, 6, and 12. The first three allow for proper motoric interaction of visual development, e.g., the coordinated movement of the eyes.

The 12th nerve allows for proper development of the tongue musculature that, at birth, constitutes the sucking reflex and later, articulation. The coordinated movement of the tongue allows for proper speech development. To further understand the influence of the midline development it is necessary to understand that it is the concerted effort of the body to maintain homeostasis. When midline muscles develop, integration takes place into the fastigeal nucleus of the cerebellum. The fastigeal nucleus of the cerebellum is involved in a closed loop of neuronal circuitry. Movement of the eyes, e.g., excites the midline nuclei 3, 4 and 6, through the muscles that they innervate about the eye, causing excitatory barrages to occur in the area acoustics. These barrages in turn excite the fastigeal nucleus in the cerebellum and increase the muscle tone of the multifidus and the intertransversaria muscles (erector spinae muscles). These neural and muscular events cause tectal spinal excitation, leading to the approximation of the facet joints of the cervical spine. These facet joints are full of

mechanoreceptors that again send information to the cerebellum as to the position of the individual's head. Cerebellar information is then transferred to the cortex for motoric reactions to the stimulus.

When aberrancies occur in the fastigeal nucleus of the cerebellum there is disruption in its closed loop. These disruptions cause only one side to excite the bilateral pathway and therefore loading takes place. Loading causes an increase in facet joint approximation on one side, and causing a greater preponderance of angulation to that side. Shifting of the head (head tilt) creates the disparity of the eyes, forcing a compensation in head position to remain level with the horizon. In early development, it is these abnormalities that create changes in the symmetry of eye movement and disparities are established.

The midline nuclei of the brainstem integrate information that causes development of the erector spinea muscles. When there is a dominance of input or excitation to an individual a disparity in proper neurologic function begins. The disparity is the greater development of one hemisphere compared to the other. The learning process receives its greatest input from the visual system. When youngsters with learning differences are examined, noticeable differences are found in hemispheric dominance.

When present, dominance has been established causing the student to learn with less than 100% of their resources. Dr. Caroline Rao, et al., The Lancet (351:1849-52). This group concludes that the altered structure symmetry seen in dyslexia is a manifestation of the abnormal development of the associated neurons or their intercellular connections or both. This group, however, has been investigating brain chemistry, which requires invasive procedures. Brain chemistry results show that deficiencies continues to exist to some degree in all individuals. While there appear to be varying degrees of learning problems, but the entire population is effected in one way or another.

Much is known concerning the ability of light to affect the stability of a person's energy, mood, sleep, concentration and the regulation of a person's circadian rhythms. Light deprivation, for example, causes fatigue, irritability, anxiety, weight gain, social withdrawal and a lack of alertness. The use of light therapy has been used to treat patients that have been afflicted with several types of brain disorders. United States Patent No. 5,562,719, issued to Lopez-Claros, describes one such method for treating seasonal affective, disorder, a condition affecting 5-20% of the population in areas of the country with decreased light levels throughout the year. United States Patent No. 5,465,939,

issued to Ochs, describes a method for assessing and amelioration of brain function after psychological and mechanical trauma. Dr. Levinson, in his world wide web site mentions successful light wave specific therapies and optometric exercises.

The human brain produces detectable signals that vary in strength and frequency over time. Human brain signals are detectable as electromagnetic waves and vary from one part of the brain to another and may, in fact, vary over time. Electromagnetic waves with different frequencies are associated with different moods and mental abilities. For example, a brain frequency of 134 Hertz or higher is know as a "beta rhythm" and is normally associated with daylight activity when all five sensory organs are functioning. In contrast, a brain wave with a frequency of 8-13 Hertz is known as a "alpha rhythm" and is associated with a relaxed creative state. Brain waves known as "theta rhythm" and "delta rhythm" have frequencies of 4-8 hertz and 0.5-4 hertz, respectively. Theta-rhythm abnormalities have been associated with learning disabilities in adolescents.

Two biochemical compounds have been implicated in the control of brain patterns and rhythms: melatonin and serotonin. Melatonin, a metabolite of serotonin, is a biochemical neurotransmitter associated with the response of

the brain to light stimuli to the eye. Dr. Rao found biochemical differences between brains of dyslexic and normal men. Attempts at changing brain biochemistry by therapeutic drug regimes are greatly limited by side effects. M. J. Koepp, in an article in the May 21, 1998 issue of Nature Magazine (393: 226-268), however, states that volunteers playing a video game or staring at a blank TV screen for 50 minutes experienced a surge in dopamine similar to levels used to treat children with attention deficit disorder. After the game ended, the dopamine levels decreased, but were still higher compared with pre-game levels.

Until recently there has been no instrument to observe and record perceptual activity of the human brain. Cortical perceptual mapping (CPM) provides researchers and educators with a powerful tool to evaluate the effect of therapeutic intervention with regard to learning and the associated behavioral problems. Using CPM it has been found that physiological blind spot mapping (PBSI) of the eye provides a window to changes in cortical perceptual mapping. Increased ability to process information leads immediately to a decrease in the size of the blind spot. CPM was used extensively to development the EYELIGHTS apparatus disclosed herein as well as developing the therapeutic regimens used for the treatment of patients.

In Figure 1, a representation of the neurological anatomy of the optical system is generally depicted as 10. Generally depicted is an eye 20 associated and controlled by the dominant cerebral hemisphere. Also depicted, is a non-dominant eye 30 associated with, and connected to, the non-dominant cerebral hemisphere. Dominant eye 20 and non-dominant eye 30 are connected via nerves 40 to the brain 50. A surface 11 is in close proximity to the dominant eye 20 and the non-dominant eye 30. The non-dominant eye 30 is stimulated by lights 12 on the surface 11 that serve to stimulate the nondominant eye 30 at a greater intensity than lights 12B that stimulate the dominant eye.

The surface 11 may be, for example, a planar or concave surface, such as sunglasses in this particular description, compared to other previously mentioned applications. The surface 11 is fitted with one or more lights 12, or using a source that can carry light such as fiber optics. In one embodiment the number of lights 12 associated with each eye is four. The number of lights may be greater depending on the needs of the patient. The lights 12 are mounted on, or integral to, the surface 11. The lights 12 may be, for example, white light, multicolored light, or plane polarized light. If the lights are multicolored, they may be primary colors or a combination of primary colors to achieve other

color combinations. The lights 12 may be turned on and off by a self-contained microcontroller 41. Alternatively, the frequency and intensity of the light 13 produced by the lights 12 may be independently powered and controlled by an operator.

The lights 12 and microcontroller 41 may be an integral part of the surface 11 and may have a self-contained power source 42. The self-contained power source 42 may be, for example, a small battery or may use a solar powered source. Alternatively, the power source 12 may be electrically connected to the microcontroller 41 and the surface 11 via wires 43.

In operation, light 13 passes through the pupil 22 of the non-dominant eye 30 and stimulates the rods and cones 24 located in the retina 25 of the non-dominant eye 30. Light 13 is reflected on each of the four known quadrants of the retina 25 depending on which light 12A is activated. By projecting the light 13 on all four quadrants of the retina 25, the light 13 and the images created on those visual fields are projected onto the retina 25 upside down and in reverse. The signal produced by the rods and cones 24 activates ganglion cell axons 27 that carry visual information from the four quadrants on the retina 25. The nervous pulses traveling through the ganglion cell axons 27 converge toward the optic disk 23 in an orderly fashion, in order to maintain approximately the same

relation to each other as they reach the optic disk 22. The visual signal is then passed along the optic nerve 28 onto the optic tract 31. The visual signal synapses into the lateral geniculate body 32 allowing cortical radiations 33 to travel back to the occipital lobe 34 before ascending to the cortical motor strip in the frontal lobe of the brain. The dominant eye is stimulated by lights 12B in the same manner, but to a lesser extent.

The apparatus and method of the present invention may be practiced using a variety of illumination intensities. In addition to different illumination intensities, different patterns and frequencies of lighting may be used to stimulate the four quadrants of the retina 25 in different manners, depending on the needs of the individual patient. It is found through examination that there are particular frequencies that are more suitable to different individuals. As well as the intensities of the light depending on ones ability to see and perceive the light. Those having very large physiological blind spots may require a greater intensity of light.

Figure 2 depicts a schematic representation of an apparatus for providing light 13 to the four quadrants of the retina 25 in order to stimulate the non-dominant cerebral hemisphere greater than the dominant cerebral hemisphere. Depicted in Figure 2 is a surface 11, in this case, on the

form of sunglasses that permit little or no light to pass through the lenses. While sunglasses are depicted, it will be understood by one skilled in the art that optical or transparent glasses that allow modified or normal light to pass through may be used. In an alternative embodiment, the glasses may be transparent, but the patient may be placed in a darkened room. Alternatively, the surface 11 may also be a monocle or an eye-patch that exclusively covers only the non-dominant eye 30. In yet another embodiment, surface 11 is a reflective surface that reflects light 13 from a light source that is not integral to the surface 11. In this embodiment, light 13 is bounced off the surface 11 onto the retina 25.

The surface 11 is depicted as having a right circle 15 and a left circle 16. Spaced evenly in each of the right circle 15 and the left circle 16 are lights 12. The lights 12 are disposed generally equidistant in the right and left circles 15, 16. The lights 12 are positioned to selectively stimulate each of the four quadrants of the retina 25.

Concomitant with, or exclusive of, the light source, sound may be selectively provided to the ear connected to the non-dominant cerebral hemisphere by a sound source that selectively provides sound to the ear connected to the non-dominant cerebral hemisphere. In most cases sound is provided to the ear that is contralateral or opposite the eye

associated with the non-dominant cerebral hemisphere that is being stimulated. The choice of sides is determined by the physician or other qualified person fitting the patient. In most cases the dominant ear needs no additional stimulation. The non-dominant ear will be the side that is stimulated bringing the non-dominant side up to a level more equal to the dominant side. In one embodiment the sound source may be part of a headset. In yet another embodiment, the headset is connected to the surface having the one or more lights.

Another embodiment of the present invention is an apparatus for treating learning disorders by selectively stimulating the non-dominant cerebral hemisphere greater than the dominant cerebral comprising, a surface placed in close proximity to a patient's eyes, one or more lights disposed on the surface, a microcontroller for controlling the lights, the one or more lights controlled by the microcontroller, wherein only the lights in front of the non-dominant hemisphere are activated, and a power source that provides electricity to the lights and the microcontroller.

The present invention provides a method for selectively stimulating the nondominant cerebral hemisphere greater than the dominant cerebral hemisphere comprising the steps of, identifying the non-dominant hemisphere of a patient and

selectively stimulating the non-dominant visual cortex of the patient greater than the dominant visual cortex using light.

In this invention a multiplicity of lights are used to stimulate the four quadrants. The quadrants include the superior and inferior nasal quadrant, and a superior and inferior temporal quadrant. In one embodiment four lights are employed with each eye. The number of lights employed, however, may be varied based on the needs of the patient.

In an alternative embodiment, illustrated in Figure 3, hemifield stimulation is employed. In this embodiment specific quadrants are excited, e.g., two or more lights may be associated with each eye. The lights of one eye, e.g., the right eye, is focused on the temporal aspects of the patient's eye. Shining the lights on one eye stimulates the nasal or medial aspect of the eye, specifically, the superior and inferior quadrant rods and cones of the retina. Once in the retina, light excites the ganglion cells before passing through the optic nerve.

Next, light decussates through the optic chiasm to pass on to the next relay that is the lateral geniculate, before finally passing to the occipital lobe area 19 in the calcarine sulcus. The left set of lights (on the medial or nasal aspects of the patients left eye) permit stimulation of the temporal fibers, superior and inferior lateral quadrant.

Stimulation follows the same pathway described above before ending in the calcarine sulcus in the occipital lobe area 19. In this pattern, the lights are focused in the inferior and superior quadrants on either the right or the left side of each eye. The neurological pathways used, will be the same as previously mentioned only that the input stimulates the side of the brain opposite the layout of the lights, i.e., lights on the right side (temporal) of the right eye and the right side (nasal) of the left eye will stimulate the left side of the brain.

An alternative embodiment of the present invention is the specific stimulation of the non-dominant auditory neurological pathway to stimulate the non-dominant cerebral hemisphere greater than the dominant cerebral hemisphere. In this embodiment differences in tone, volume and the type of auditory signal is are varied to achieve the desired effect.

In operation, a sound source, such as those found on a pair of headphones, is positioned in a patient's head and the output from the headphones is directed solely to the ear associated with, or connected to, the non-dominant cerebral hemisphere. Alternatively, the sound source may be, for example, an individual speaker that is hooked on to the ear, or is held by a strap or headset. In yet another embodiment, the sound source 44 may be attached to the surface 11. In

practice both ears are actually stimulated. One ear is associated with an ear piece that would play some sort of stimulating noise. Where as the opposite ear would only be stimulated from the normal sounds of the environment. The non-dominant hemisphere is related to the ear receiving extra stimulation from an auditory device.

The volume and type of sound produced by the sound source positioned over the ear to be targeted may be controlled by the user, or by an operator via a remote connection. The type of sound may be any of a range of types of musical selections, varying from classical to jazz to the use of binomial sounds and beats. Alternatively, sounds that are not human generated may be used, such as those found in nature. Examples of sounds found in nature include, but are not limited to, ocean surf, falling rain, forest sounds and the like.

The volume and type of sound generated by the sound source over the target ear may also be varied. The sound generated by the sound source stimulates the auditory pathways to the non-dominant hemispheres of the brain. The volume may be set at a constant level throughout the treatment, or, may be varied during the treatment according to the individual patient's needs and diagnosis. As with the apparatus connected to the patient's eyes, the sound sources may be self

powered, or may derive their sound and power from an independent source.

In another embodiment of the present invention, the visual and auditory stimuli to the non-dominant hemisphere may be used separately but in a serial manner. Alternatively, they may be actuated concurrently, depending on the patient's individual needs.

The present invention provides a solution to the problems of individuals that have to deal with distractions on a daily basis. These distractions take away from their optimal mental health. The present invention permits individuals with internal or external distractions to focus their mental performance and their ability to perceive information. Performance and the ability to perceive and retain information is critical to those with learning disabilities, or with a need for enhanced learning abilities. The present invention is inexpensive, thereby helping those who have financial and educational needs to afford a device that selectively increases learning potential.

The ability to focus is also critical to those in the academic, business or athletic world. Using the device of the present invention, the visual and/or auditory neurological pathways of the non-dominant cortical hemisphere of individuals can be stimulated to cause excitatory post

synaptic potentials (EPSP). Selectively stimulating the non-dominant cortical hemisphere to cause collateral synaptic activity of the visual and/or neurological pathways helps to achieve the goals of optimal mental health, to focus performance and to focus the person's ability to perceive and retain information.

While the visual and auditory stimulation apparatus have been described separately, one of skill in the art will know that devices may be used concurrently. When two devices are used they may be part of a single unit, and therefore, may share the microprocessor 41 and power source 42. Alternatively, the devices may be separate and derive their power from the same source, or from independent power sources 42.

Using the light therapy system of the present invention, as shown in part in Figure 1-3, it was first demonstrated that the apparatus could be used to increase the learning and athletic potential or participants by changing and controlling critical brain activity of the non-dominant cerebral hemisphere in relationship to the dominant cerebral hemisphere. Next, the potential of stimulating the non-dominant cerebral hemisphere by light therapy was used to stimulate and impact the participant's mental and physical performance and well being. The apparatus and methods

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disclosed herein were then used not to enhance the performance of individuals in generally good health, but to treat patients with disease conditions.

The three children documented below had severe learning and behavioral problems when referred to the inventor's clinic. During consultation and subsequent therapeutic treatment of each these of young patients, the it was found that there is a distinct correlation between a strongly dominant cerebral hemisphere and learning problems. In each case, therapeutic intervention with EYELIGHTS had a dramatic effect on the students learning capabilities and disciplinary problems in the classroom.

All three children had similar backgrounds. Two came from stable environments with both parents married and living together. The third came from a divorced, yet remarried, mother and appeared to be now in a stable environment. All children were healthy and well-nourished. Another common feature was that these patients were very particular in their culinary tastes. Each was found to have a strongly dominant hemisphere.

Case # 1: The patient was a boy of 6 years, 11 months. He was a very poor student after two years in kindergarten and has been socially promoted from kindergarten to the first and then to the second grade. He could not write his ABCs and had

dyslexic symptoms. He was referred to therapy as the public schools had been unable to resolve his problems. The decision was made in conjunction with his parents to try light therapy using a prototype of the EYELIGHTS. Immediately prior to the installation of the glasses for the first time, the patient was asked to write his name on the chart. Figure 4 is a writing sample from the patient prior to exposure to the present invention. The EYELIGHTS glasses were then placed on his head specifically to address his problem and he was immediately asked to write his name. Figure 5 shows the results in writing ability after use of the present invention.

As seen above, the therapeutic regimen had a dramatic effect on the patient's pre and post ability to write his name. In Figure 4, the student wrote his name in a mirror pattern from the right side of the paper to the left, a typical dyslexic symptom. The name shown in Figure 5 was written immediately after the EYELIGHTS therapy was used to stimulate the non-dominant hemisphere. The student was able to write his name correctly from left to right and the letters were in the correct relationship to the baseline. The patient received three months of therapeutic treatment with one session every two weeks. His mother reports that he no longer has dyslexic symptoms, his reading and writing are improving

at a rate that will bring up to his class level very soon and has no discipline problems in the classroom or at home.

Case # 2: A boy of 10 years, 9 months old experienced difficulty focusing on his work and completing tests and schoolwork in a timely manner. He was consistently in trouble and behind the rest of class due to his inattention and unwillingness to partake and complete tasks at school as well as homework. During the examination, the patient was given a book from which to read in order to quickly assess his cognitive ability to learn. When asked to recite what was read he demonstrated virtually no ability to recall. Intervention with the EYELIGHTS was given and another area of the book was then read. The patient's ability to read, and his recall of the information, improved significantly. The patient also received three months of therapy with one session every two weeks.

At this time, the patient's mother and teacher report that the patient is at the top of his class and his confidence level increased along with his ability to finish and understand the task at hand.

Case # 3: A boy of 6 years, 11 month old. He experienced behavioral problems, learning disorders and hyperactivity. In school he was very disruptive and was not able to sit in his chair and pay attention, or he continued to

talk and upset and disturb those around him. The patient's reading was found to be from right to left and was dyslexic in his ability to write. The patient also received three months of therapy with one session every two weeks.

Post therapy changes have been significant for the patient in case study #3. The patient no longer exhibits dyslexic symptoms, presently reading and writing while staying up with the rest of his class. The patient now is able to sit in his chair and partake of classroom activity in a normal fashion.

The applications of this therapy are far-reaching. These case studies indicate that the symptoms of ADD and dyslexia may be ameliorated very quickly and at a moderate cost without chemical intervention. The therapy and the hardware may be offered through commercial learning centers, neurologists, kinesiologists, optometrists, medical doctors, psychologists and other groups with the necessary scientific training and license to perform the necessary evaluations diagnose the problem and prescribe the therapy. Government applications are also significant. For example, the therapy may be useful for enhancing military training, performance, endurance and cognitive abilities. The apparatus may also find use for the enhancement of training, resulting potentially in a reduction

of training time and therefore a significant reduction in training costs.

The implementation of EYELIGHTS for this type of light therapy causes an excitation of the non-dominant hemisphere, both cerebellar and cortical and to a lesser extent to the dominant hemisphere already getting the greatest amount of the daily educational workout. The type of excitation used to implement the therapy uses a feed-forward or efferent-copy mechanism. The process complements the already dominant hemisphere with an elevation of the non-dominant hemisphere, thereby elevating the entire neuraxis.

The results obtained using the present invention may be documented scientifically as follows. A Visual Evoked Potential (VEP) measurement allows an observer to measure the speed at which the brain reacts to an external light stimulus. A VEP is comprised of three parts. Surface recording electrodes are strategically along the midline of cranium at frontal center (Fz), parietal central (Pz), and occipital central (Oz) locations. A stimulus in the form of a red and black checker board flash pattern is applied. The interaction between the perception and tile realization of light to the neuraxis is measured. The test will measure hemifield stimulation to the right and the left eyes and the test will be performed with and without the EYELIGHTS in place.

A trained observer, e.g., the patient's teacher or counselor, documents the patient's behavioral and learning tendencies at the beginning of the evaluation and therapy period. The observer establishes a baseline condition for each student both with and without the EYELIGHTS. A three-month daily therapy routine for each student will be initiated. The observer documents changes in observed behavioral and learning tendencies at intervals, e.g., weekly, during the therapy period. At the end of the therapy the observer performs an identical set of PBSI and VEP tests with and without the EYELIGHTS to establish changes from the original tests. The PBSI and VEP data and teacher observations will be correlated.

Further studies may be conducted as follows. Twenty or more students are selected from the surrounding community school systems. The students include children in elementary school, grades 1-6. To be selected the student must have been clinically diagnosed to have ADD, ADHD, or dyslexia by a child or school psychologist and the student's counselors. The parent or guardian of each child will be required to sign a consent form before entering the program. The children are split equally into a test group and a control group. It is hoped that an equal number of boys and girls may be included in the groups.

The observer will meet with the trained child or school psychologist to generate a standard observation protocol. The protocol is used during the evaluation and study periods to establish baselines and document progress and final state conditions for each student. Each student receives a thorough neurological and orthopedic examination and the results recorded. Any student with a previous history of epilepsy, migraine or seizures will be excluded from this test program.

The protocol may include, e.g., a PBSI (physiological blind spot indicators) test to determine right and left hemispheric deficiencies. Also, a baseline VEP test is performed on each student. These techniques will establish the baseline electrical activity of the brain. Three electrodes are placed along the midline of the cranium or sagittal suture. The electrodes are placed at the following locations Fz (frontal lobe center) Pz (parietal lobe center), and Oz (occipital lobe center). The measurements are gather for the interaction between the perception and the realization of light to the individual students neuraxis. Five separate brain stimulations may be performed; bilateral stimulation, right hemifield stimulation, left hemifield stimulation, right monocular stimulation, and left monoccular stimulation. This information is recorded and used to support the use of light

stimulation in an individual defined with a specific cortical lesion.

Next, each student is fitted with a pair of EYELIGHTS with the correct monoccular stimulation per the results of the testing in 4) and baseline data with tile glasses will be taken as disclosed hereinabove. Following the determination of basal brain activity, the therapy period begins. Each of the students in the therapy group, under the supervision of his teacher or counselor, will be asked to wear the EYELIGHTS for a period of 5 minutes at the beginning of each school day, after lunch, and at the end of the school day. The test period may be, e.g., three months. The teacher or counselor is asked to record that the student actually completed each step in the therapy.

At the end of the therapy period each student in the therapy group and the control group will be tested as disclosed hereinabove using the protocol determined or used for that patient. During the therapy period, the teachers and counselors involved with both the therapy and control groups will be asked to document the classroom performance of each child in the study oil a weekly basis. At the end of the testing period, the test results and the observation reports are tabulated and analyzed. The correlation between the PBSI and VEP test for each patient are then established.

The following is a list of equipment that may be used to obtain the PBSI, VEP and other measurements disclosed in the protocol hereinabove, and include:

1) a Cadwell Sierra 11 Visual Evoked Potential;

2) a Physiological Blind Spot Mapper;

3) a Micromedical Technologies Real Eyes infrared frail camera and goggles;

4) a video recorder/television

5) a 8mm video camera/VCR.

Based on the observations made with the patients in the three case studies a physiologic mechanism is proposed for the observations made using the present invention. In no way should the proposed physiologic mechanism be used to limit the scope of the present invention. It has been observed that the pontine epinergic cells are important for human survivability in regards to limbic reality because it provides epinergic neurotransmitters that drive the dopaminergic A10 cells and the superior and inferior colliculus; resulting in stimulation of the lateral and medial geniculate bodies. These thalamic nuclei fire projections to primary association areas in the temporal lobe, parietal lobe, and area 17 of the occipital lobe.

This portion of the limbic system is important for human survival because it is responsible for firing the rostral

reticular activation system of the mesencephalon that activate the cortex. Pontine epinergic cells allows the tectum to reach summation quickly, thereby informing our cortex about impending danger from the modalities light and sound (i.e., via the superior/inferior colliculus) but also it provides epinephrine for the receptors of the right limbic expression.

The right brain has a higher density of epinergic receptors than does the left brain, which is higher in dopaminergic receptors. Without proper activation of the epinergic centers a patient may experience a loss of primary humanistic drives (i.e., to eat, get up and go to work, have sex, etc.).

The amygdala is also rich in dopamine and epinephrine. In order to ascertain which system is responsible for decreased activation of the amygdala, it is necessary to compare whether the amygdalal response is associated with the dopaminergic (mesencephalic) or epinergic (pontomedullary) system. To make the distinction, it is necessary to examine the cranial nerves associated with the specific regions of the brainstem. It has been found using the apparatus and the method of the present invention, that if a patient has a decrease in epinergic activity resulting in an amygdala response or decreased pontine firing, the patient could suffer form a pontine/cerebullar type of dysfunction.

Therefore, using the present invention, it has been found that if one decreases the firing of the cerebellum, there is a high probability that there is a decrease in the activity of the pons and of the integration into the Locus cerulcus and the sub ceruicus. Decreases in mesencephalic activities of, e.g., Ed-Wes, substantia nigra, red nucleus, CN's 3,4 etc. would be indicative of dopaminergic dysfunctions. One way to increase A10 firing is to increase the FOF of neurons in the vicinity. That is the trochlear nucleus in the caudal mesencephalon. To do so one requests that the patient move their eyes down and in, towards the side of the lesion.

Epinergic systems also descend caudally and evoke stimulation to the dorsal and medial raphe nuclei and the pontomedullary junction directly increasing their FOF without going to the brain. These systems serve to attenuate secondary stimuli and allow primary stimuli (food, sex, etc.) to be pleasurable. The descending portion of the epinergic system is serotonergic and descends within the pontomedullary inhibitory projects within the ventral lateral funiculus of the cord ipsilaterally. This is the same projection that: 1) actively inhibits inhibition to the ipsilateral alpha and gamma motor pools; 2) actively inhibits the IML; and 3) actively inhibits the ipsilateral anterior compartment muscles about T6 and post compartment below T6.

When a patient loses the ability to activate this system, there is a probability that secondary stimuli may reach cortical representation. The symptomatology may present as day dreaming, states of confusion, and in some cases even suicide. It is well known that a percentage suicide victims have been shown on autopsy to have decreased levels of 5-hydroxytryptophan (5-HT, a serotonin derivative) that would be produce as a consequence of epinergic firing of the Raphae nuclei of the brainstem. When epinergic firing of the Raphae nuclei is decreased, the attenuation of stimuli also decreases and the cortex is subject to an increase in somatotopic representation that exceeds metabolic rate. Hence, potential suicide. Using the cerebellar stimulation method and apparatus of the present invention control over (feedforward, feedback, and efferent copy) cranial nerve stimulations is shown herein to help control limbic dysfunction's.

As already mentioned, the amygdala is an area that is very important to human survival. Children have limited integration into the amygdala from the frontal lobe due to a lack of cortical plasticity. It is a loss or lack of the inhibitory mechanism from the frontal lobe that causes kids to "say and do the strangest things" that most adults would not. A child builds plasticity within the integrating pathways to the amygdala either from physical stimuli through cerebellar

feedback pathways or the comforting feedforward pathways from a mother's love. The integration and expression of the amygdala into the limbic system depends on so many factors that doctors in general, only make predictions of how and why things developed the way they did.

Reference is now made to Figure 5, in which a schematic diagram of a system 100 is shown in accordance with one embodiment of the present invention. System 100 includes a microcontroller 102 that controls various functions of the circuit 100, including sending and receiving electrical signals. Microcontroller 102 includes programmable flash memory for, e.g., storing code. Microcontroller 102 may typically be a conventional device such as, for example, a AT90S1200 microcontroller manufactured by Atmel, or other such commercially available devices.

Microcontroller 102 sends electrical signals through resistors 103 to light sources 104A and light sources 104B (collectively, light sources 104) via electrical interfaces 105. Light sources 104 may be conventional light emitting diodes (LEDs). In the embodiment depicted in Figure 5, in which the number of light sources 104 is eight, four light sources 104A may be associated with a patient's first eye, and four light sources 104B may be associated with a patient's second eye; however, as mentioned previously, it should be appreciated by one skilled in the art that the number of light

sources 104 may be varied according to the requirements, diagnosis and preferences of the patient.

In the embodiment shown in Figure 5, port B of microcontroller 102 is an 8-bit bidirectional I/O port that provides current to light sources 104. When pins of port B are used as inputs and are externally pulled low, they will source current if the internal pull-up resistors are activated. It should be understood by one skilled in the art, however, that an active high device may be employed consistent with operation of the present invention.

Port pin 106 of microcontroller 102 functions as a ground pin (GND). Port pin 108 of microcontroller 102 functions as a supply voltage pin (VCC). Figure 5 depicts a battery 110, which may be approximately 3 volts, as a suitable supply voltage. It should be appreciated by one skilled in the art that the present invention is not limited to the described supply voltage but is intended to encompass a broad range of supply voltages according to the specifications of microcontroller 102.

Port pin PB5 112 of microcontroller 102 has an alternate function as a data input line for memory downloading (MISI). Port pin PB6 114 of microcontroller 102 has an alternate function as a data output line for memory uploading (MISO). Port pin PB7 116 of microcontroller 102 has an alternate function as a serial clock input (CLK).

A low level on Reset pin 118 of microcontroller 102 for a certain time period, for example, longer than approximately 50 ns, will generate an external reset. An external interrupt may be triggered by setting port pin PD2 (INT0) 120 of microcontroller 102. As shown in Figure 5, an on/off switch 122 is connected to port pin PD2 120. A switch 124 is connected to port pin PD0 126 of microcontroller 102, and functions as a mode selector, rotating among the available modes.

The intensity of each of light sources 104 may be increased or decreased as desired by making adjustments in duty cycle. For example, microcontroller 102 may pulse one of light sources 104 such that it is on for 10 ms, and then off for 30 ms. To achieve a higher intensity, microcontroller 102 may pulse one of light sources 104 such that it is on for 30 ms, and off for 10 ms.

Figure 6 depicts a rear elevation view of a device 150 employing the features of system 100. Device 150 includes a first frame 152, lenses 154 mounted therein, temples 156 connected to the first frame 152 via hinges (not shown) and a nose piece 158. Attached to the first frame 152 is a second frame 160, on which two upper left LEDs 162, two upper right LEDs 164, two lower left LEDs 166 and two lower right LEDs 168 are mounted. Further included in device 150 are a power switch button 170, a mode selector switch button 172 and a

battery receptacle 174. The second frame 160 may be removably attached to the first frame 152 via snaps, for example.

To stimulate the temporal area of a patient's brain, where learning and memory functions are located, if the patient's right eye is dominant, the power switch button is depressed until a set of LEDs are illuminated. The mode selector switch button 172 is then depressed until the upper left LEDs 162 are flashing brighter than the lower left LEDs 166. In order to stimulate the temporal area of a patient's brain, e.g., where the patient's left eye is dominant, the power switch button is depressed until a set of LEDs are illuminated, and the mode selector switch button 172 is then depressed until the upper right LEDs 164 are flashing brighter than the lower right LEDs 168.

To stimulate the parietal area of the patient's brain, where sensory and motor functions are located, if the patient's right eye is dominant, the power switch button is depressed until a set of LEDs are illuminated, and the mode selector switch button 172 is then depressed until the lower left LEDs 166 are flashing brighter than the upper left LEDs 162. In order to stimulate the parietal area of a patient's brain, e.g., where the patient's left eye is dominant, the power switch button is depressed until a set of LEDs are illuminated, and the mode selector switch button 172 is then depressed until the lower right LEDs 168 are flashing brighter than the upper right LEDs 164.

Reference is now made to Figure 7, which depicts a flow diagram of the process steps that may be executed by the code embedded in microcontroller 102 in accordance with one embodiment of the present invention. Process flow starts at step 200, and a power check is initiated in step 202. If the power is on, process continues to step 204, in which the Non Volatile RAM is checked to see if a particular mode was set during a previous run. If there is no previous set state, the system is powering on for the first time. If the system 100 was powered on previously, a mode will be stored in the Non Volatile RAM and automatically read into data registers.

Next, in step 206, the voltage of VCC at port pin 108 is read, and the internal oscillator of microcontroller 102 may be adjusted accordingly. In steps 208 and 210, the appropriate light sources are turned on. The light sources are then pulsed in step 212 such that they are on for a certain length of time and off for a certain length of time pursuant to a prescribed duty cycle.

The mode switch is checked in step 214, and if the mode switch is set to a particular value, then process flow continues to step 216. After an appropriate amount of clock cycles, all light sources are turned off. Otherwise, the process flow reverts to step 204.

In step 218, a counter is incremented and then compared to a predetermined value in step 220. If the counter is less than that value, then process flow loops to step 208; if the

counter is equal to the predetermined value, then process continues to step 222. Microcontroller 102 then sleeps for an appropriate number of cycles in step 222, and time is checked in step 224 to determine whether the microcontroller 102 should continue sleeping, or whether process loops back to step 204. Code for implementing this process is shown in Appendix I.

APPENDIX I

```
;   TC0 runs with prescale=1. no reloading necessary.
;       interrupt frequency is approx 4KHz.  LEDs are
        brightness controlled by being on for (between 1
        and 16) out of every 16 ticks, reducing frequency
        to approx 250Hz.
;
;   Brightness control works as follows:
;       At the BEGINNING of a brightness control cycle, if
        the Flash Duty Cycle counter says that the LEDs
        should be ON, then both the High and Low Intensity
        LEDs are turned on.
;       After the appropriate amount of time, the Low
        Intensity LEDs are turned off to perform Brightness
        control.  Later, the High Intensity LEDs are turned
        off.  Even later, the Duty Cycle count point is
        reached, and the Brightness Control Cycle begins
        again.

;   LEDs are driven thru separate resistors: LOW=ON.
;       All LEDs are on Port B to simplify software.
;
;   Button Functions:
```

```
;       One button serves as the power control.  It is tied
        to PD2, so it can wake up the AVR from full power-
        down mode by using a low-level int.  The other
        button rotates between the available modes.

;   Button denounce method:
;       When pressed, action occurs immediately.  Debounce
        action comes into play by waiting a defined time
        before re-checking the switch to see if it has been
        released yet or not.

;   Watchdog:
;       The watchdog is used to wake up the processor to
        check for button presses.  It is also used to do
        the 20-second OFF time while running.
;
;   RESET-based startup conditions:
;       OFF20:  dec ctr.  If  =0, go to ON20 mainline
                handler to restart 20-sec flashing.  If
                not =0, check for on/off button.  If
                seen, do powerdown lights & debounce
                button, then go into Wdog sleep
                condition.  If button not pressed, go
                into Wdog Sleep.
```

```
;           POWERUP:  init variables, go into IDLE mode.  Don't
                      check buttons.  Also, kick all lights on
                      Full Intensity
;           IDLE:     check for On/Off; if seen, recall stored
                      EE LED mode, force onto lamps while
                      On/Off button remains pressed.  After
                      release & debounce, go into ON20 mode.
;           ON20:      If this condition should occur, do IDLE.

;     INTERRUPT-based conditions:
;           ON20:     timer tick runs first, then RETIs into
                      mainline.  Mainline checks for On/Off &
                      Mode button; if none, goes back into
                      Idle-sleep.
;           IDLE:     in this condition, just output the
                      current LED state as defined in HiLights
                      & LoLights, without doing any 7.77Hz
                      flashing.  This is used at power-down as
                      well as mode-change and powerup.

.include "1200def.inc"

.equ MAINCLOCK =    170000    ;CPU clock (either internal or
                              ext.)
```

```
.equ FLASHRATE =    777         ;LED main blink frequency,
                                TIMES .100

.equ WATCHDOG  =    MAINCLOCK ; (internal RC osc on AVR chip)
.equ WDOGRATE  =    (WATCHDOG / 1024) / 128       ;
                    (frequency)

.equ OnSeconds = 4              ;# of secs that LEDs blink
                                while running
.equ OffSeconds     = 7         ;# of secs they're BLANK while
                                running

;This number is the total number of timer ticks in one
brightness control period.
.equ DutyBright     = 16

;This setting is the number of ON-ticks for a HIGH-brightness
LED
.equ HighBright     = 16 ;(allowable range is from 1 to
                          DutyBright)

;This setting is the number of ON-ticks for a LOW-brightness
LED
```

```
.equ LowBright      = 3  ;(1 is the minimum allowable
                         setting)
.equ ModesInUse     = 8  ;set this to 6 if using modes 0-5,
                         or etc.

;Customize for each desired pattern.  Any LED not included
will be OFF.
.equ Pattern0H = 0x90    ;adjust these to fit mode selection
.equ Pattern0L = 0x60
.equ PatternIH = 0x60
.equ Pattern1L = 0x90
.equ Pattern2H = 0x09
.equ Pattern2L = 0x06
.equ Pattern3H = 0x06
.equ Pattern3L = 0x09
.equ Pattern4H = 0x81
.equ Pattern4L = 0x42
.equ Pattern5H = 0x42
.equ Pattern5L = 0x81
.equ Pattern6H = 0x18
.equ Pattern6L = 0x24
.equ Pattern7H = 0x24
.equ Pattern7L = 0x18
```

```
;-----------------------------------------------------

.def SaveSreg       = r0      ;holder for SREG during ISRs
.def Alive0         = r1      ;is it running presently?
.def Alive1         = r2
.def Alive2         = r3
;3912Hz rate src (1MHz. /256)
.def BrightCtr      = r16     ;counts sets of 16, is COMPARED
                              AGAINST.
.def FlashCtr       = r17     ;to time the 7.77mS flash rate
                              & duty cycle
.equ DutyFlash =
                (((MAINCLOCK/256)*100)/DutyBright)/FLASHRATE)
.equ OnFlash        = (Duty Flash / 2)        ; if DutyFlash
                                              is odd, make ON
                                              short
.equ OffFlash       = DutyFlash - OnFlash     ; allow OFF to
                                              be the longer
                                              one
;DutyFlash          = 125 if CLK=4MHz    ; =31 if CLK=1MHz;
                                        =11 if CLK=350KHz
.def Ctr20          = r18           ;used for timing the 20-
                                    sec interval
```


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```
.equ On20sec        = OnSeconds*FLASHRATE/100     ;how manv
                    (7.77Hz) flashes to do
.equ Off20sec  = OffSeconds*WDOGRATE    ;how    many    Wdogs
                                        occur during OFF
.equ WDOGSETTING = 0X0B  ; = approx. 0.125sec (is the /128
                         setting)
.def OpMode     = r19
.equ POWERUP    = 0x00   ;(RAM is cleared at initial power-
                         on)
.equ IDLE       = 0X01   ;in an inactive mode
.equ OFF20      = 0x02   ;timing the 20-sec OFF period
.equ ON20       = 0x03   ;doing the 20-sec ON period

.def KeyState   = r20    ;which key being pressed
.def KeyCount   = r21    ; how many sets of 10mS for debounce
.def TickCtr    = r22    ;inc this every tick; misc usage.
.def LEDMODE    = r23    ; hold LED pattern selection (a copy
                         of EE)
.def HiLights   = r24    ; hold bit positions of current Hi-
                         Intens LEDs
.def LoLights   = r25    ; hold bit positions of current Lo-
                         Intens LEDs
.def ISR0       = r28    ; reserved for use by ISR only
.def temp2      = r29
```

```
.def templ      = r30      ;used by mainline
.def temp0      = r31
.def ZL         = r30
.def ZH         = r31

.equ ButtonPin = PIND
.equ ONOFFSW   = 2
.equ MODESW    = 0
.equ MaxModes  = 4         ;this defines max possible # of
                           modes.
;================================================
.org 0
RstVec: rjmp        Startup
TR00: rimp          OnOffButton          ;PD2, Ext. Int. 0
                                         (wakeup)
;the following NOPs are placeholders for INT vectors on 8515,
so the code will run on both the 90S1200 & the 90S8515 w/ no
changes.
     rimp Timer0                ;INT1
     nop                        :Tlcapt
     nop                        ;TlcompA
     nop                        ;TlcompB
     nop                        ;Tlovf
```

```
Timer0: ;this is the (approx) 4KHz timer tick
        in   P0.SREG
        inc  TickCtr          ;misc usage
        inc  BrightCtr        ;bump Brightness duty cycle
                              counter
        cpi  BrightCtr.DutyBright     ;at end of Bright
                                      Control Cycle?
        brne DoBrightness     ;if not, check for Low/Hi steps
        clr  BrightCtr        ;reset the brightness control
                              counter
    ;Completed one full cycle of brightness duty-cycling now.
        cpi  OpMode.IDLE      ;if IDLE mode, don't turn off
                              LEDs!
        breg LightsOn
    ;not IDLE mode, so go ahead and FLASH the lights.
        inc  FlashCtr;bump the Flash Rate timer
        cpi  FlashCtr.DutyFlash  ;at end of Duty Cycle
                                 period?
        brne      ChkOffFlash    ;if not, see if in OFF
                                 time of 7.77Hz
        clr  FlashCtr
    ;Done      with one 7.77Hz period now.
    ;Turn ON LEDs f not at end of 20-sec ON-period.
        inc  Ctr20
```

```
            cpi  Ctr20,On20sec
            brne LightsOn; 20-sec ON time still going!
        ;need to drop into 20-sec OFF time now
            ldi  Ctr20.Off20sec ;set up Wdog tick count
            ldi  OpMode,OFF20   ;set new mode!
WDSLEEP:
            cli                 ;kick the dog!
            wdr
            ldi  ISR0,WDOGSETTING
            out  WDTCR,TSR0
;           ldi  ISR0.0x00      ;shut off ext int
;           out  GIMSK.ISR0
            ldi  ISR0.0x30      ;PowerDown mode. w/ low-level
            out  MCUCR.ISRO     ;external interrupt mode.
;           ldi  ISR0.0x40      ;Enable ext int!
;           out  GIMSK,ISR0
;           sei                 ;Enable all ints
            sleep                    ;we'll get a Reset from
                                WDT
;           nop
            rjmp Startup ;this is here JUST IN CASE ...

ChkOffFlash:
```

```
            cpi   FlashCtr,OffFlash   ;if at or above this
                                       count.
            brsh      HighOff;        ;don't turn the lights
                                       on!
LightsOn: ;turn on LEDs according to current Mode
            mov   ISR0.HiLights  :grab bits for Hi-Intensity
            or    ISR0,LoLights  ;merge bits for Lo-Intensity
            com   ISR0
OUTLED:     out   PORTB.TSR0
RTI: ;go to Main to check Mode button, then go into Idle mode.
            in    R0,SREG
            reti


.DoBrightness: ;check for when to turn OFF the LED banks
            cpi   BrightCtr,LowBright ;shut off some yet?
            breg LowOff
            cpi   BrightCtr,HighBright
            brne RTI
HighOff: ;turn off Hi-Intensity lights now
            ldi   ISR0.0xFF       ;just turn ALL LEDs off
            rjmp OUTLED


PwrTbl: ;constant sequence for power management
            .dw   0x3572,0x2165,0x4F43,0x5950
```


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```
            .dw  0x4952.0x4847.0x2054,0x3931
            .dw  0x3939.0x5220.0x4720.0x4620
            .dw  0x4952,0x5345,0x4423,0x3700
LowOff: ; turn off Low-Intensity lamps now
            in   ISR0.PORTB      ;read LED status
            or   ISR0.LoLights   ;force ONLY the Lo's HIGH (off)
            rjmp OUTLED

;================================================


Startup:        ;check if were alive before got reset
            ldi  temp0, 0x80          ;turn off comparator
            out  ACSR,temp0           ; to save power

            ldi  temp0,0xFF           ;set up stack for 8515
                                      (test only)
            out  0x3D.temp0
            ldi  temp0.0x01
            out  0x3E,temp0

            ldi  r29,0x52 ;set up a unique string
            ldi  r30,0x47 ; that would NOT be present
            ldi  r31,0x46 ; at initial powerup...
```


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```
        cp   r29,Alive0     ;Is the string present in RAM?
        cpc  r30,Alivel
        cpc  r31.Alive2
        breq Resume     ;Jump if string already there!

;At this point, we've not been awake before.  Init the
chip, see if a button is pressed and handle it, or go
into SLEEP mode with On/Off button interrupt armed.

ClearM: ldi  ZH,0       ;clear memory before proceeding
        ldi  Zl,30
ClrMem: dec  ZL
        st   Z.ZH
        brne ClrMem     ;if Z > 0, loop

        ldi  temp0.0x52     ;init unique-string
        mov  Alive0,temp0
        ldi  temp0.0x47
        mov  Alivel.temp0
        Idi  temp0,0x46
        mov  Alive2,temp0

Resume:  :after  Reset,  must  ALWAYS  re-init  ports  &
peripherals
```

```
            ldi   temp0.0xFF          :init the LED port
            out   PORTB.temp0     :force all LEDs off
            out   DDRB,temp0          ;set dir to OUTPUT

            out   PORTD,temp0     ;turn on ALL pullups on PORTD

;-------------------------------------------------------------
-------
OK:
;Now determine currrent RESET mode & react accordingly.
     ;check if timing the 20-sec OFF period
          cpi   OpMode.OFF20
          brne ChkModel
     ;we ARE in OFF20 mode.
          dec   Ct420
          breg DoneOFF20       ;just finished OFF time, so go
                               wake up!
     ;Still in the 20-sec OFF period, but check for ON/OFF
     button!
          sbjc ButtonPin.ONOFFSW
          rjmp WDSLEEP         ;go to sleep again

     ;ON/OFF button is pressed, so kick on all lights until
     released.
```


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```
        ; and then go into WDOG SLEEP mode (or NO-WDOG SLEEP if
        use PD2).
PwrOff:   ldi  TickCtr,(256-40)    ;approx 10mS debounce
PwrOff1:
          ldi  OpMode.IDLE
          clr  LoLights; force all LEDs to high intensity
          ser  HiLights
          rcall     StartTimer
          sei                 ;global enable ints
PwrOffLoop:
          cpi  TickCtr.0
          brne PwrOffLoop          ;wait here for 40 ticks
PwrOffLoop2:
          sbis ButtonPin.ONOFFSW   ;stop looping if released
          rimp PwrOffLoop2
          cli
          ldi  temp0.0xFF          ;turn off LEDs
          out  PORTB.temp0
          rjmp WDSLEEP   ;enter  SLEEP,  in  IDLE  state,  no
      button!
```

```
DoneOFF20:     ;have lust finished timing the 20-sec OFF
period.
     ;start up the Timer again now
          rcall StartTimer
;    ;set up the On/Off button interrupt
          ldi  temp0.0x00          ;shut off ext int
          out  GIMSK.temp0
          ldi  temp0.0x22          ;SLEEP='IDLE'     .     w/
                                   falling-EDGE
          out  MCUCR.temp0    ; interrupt sense
          ldi  temp0.0x40          ;enable ext int
          out  GIMSK.temp0
     ;set new Operating Mode
          ldi  OnMode,ON20    ;assert new mode status
     ;watch the Mode & On/off buttons from now on...
          rjmp             CheckButton

;-------------------------------------------------------------
---

ChkModel: ;next, check if we lust had initial power-up
          cpi  OnMode.POWERUP ;did we just power up?
          brne ChkMode2
     ;force into IDLE State, but kick on lamps BRIEFLY first
```


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```
            ldi  TickCtr,1      ;approx 62mS
            rjmp PwrOff1

;-----------------------------------------------------------
---

ChkMode2: :if IDLE, waiting for ON/OFF button press to
activate!
            cpi  OpMode,IDLE
            brne ClearM                         :got a completely
     unknown reset
            ;woke up to check for ON/OFF button press
            sbic ButtonPin.ONOFFSW
            rimp WDSLEEP        ;no button, so go back to sleep!
            ;ON button pressed, so restart previous mode
            rcall     GetLEDmode
            rcall     SetUpLEDs
            ldi  Ctr20.0
            ldi  OpMode.ON20
            rcall     StartTimer
            ldi  KeyState.1     ;set up to debounce PowerOn
condition
            ldi  KeyCount.60
```


607

```
        ;now go into light-sleep mode, then loop waiting
        for either
        ;a mode change or a power-off request.  Whenever we
        power off, we must save new MODE value to EEPROM if
        it changed during operation.  This can be
        determined by checking current mode against what's
        in EE, and if different, store the current.

CheckButton:    ;this monitors the Mode button after a Timer
Tick

        ldi  temp0.0x22     ;Idle Mode, failing-EDGE ext int
        out  MCUCR,temp0
        sei                 ;enable ints again
        sleep
        nop
    ;check button denounce first
        cpi  KeyCount,0     ;are we debouncing?
        breg CBonoff
    ;we ARE debouncing, but which one?
        cpi  KeyState,1     ;is it power-on?
        brne DBMode
    ;we are debouncing the On/Off button
        sbis ButtonPin,ONOFFSW
```


CAS

```
            ldi  KeyCount,60    ;restart 15ms timer
            dec  KeyCount
            rjmp CheckButton    ;always loop back now.

DBMode:                         ;debouncing the MODE button
            sbis ButtonPin.MODESW
            ldi  KeyCount,60    ;restart 15ms timer
            dec  KeyCount
            rjmp CheckButton    ;always loop back now.

Cbonoff: :check On/Off button first!
            sbic ButtonPin,ONOFFSW
            rimp CBMode
      ;handle power-down now
            rcall     SaveMode ;save LEDMODE if needed
            rjmp PwrOff        ;wait for button release too

      CBMode: ;check the MODE button now
            sbic ButtonPin.MODESW
            rimp CheckButton    ;loop back if not pressed
      ;button is pressed.
            clr  KeyState
            ldi  KeyCount,60    ;15mS
            inc  LEDMODE        ;bump and
```

```
            andi LEDMODE.7
            cpi  LEDMODE,ModesInUse ;check against max used
            brlo CBModel
            clr  LEDMODE
CBModel:
            rcall     SetupLEDs
            clr  Ctr20
            rjmp CheckButton    ;loop back


;This can be called from one of three means;
;     1) during 20-sec ON-time of LEDs, via EDGE interrupt
;     2) during 20-sec OFF-time, via LOW-LEVEL int
;     3) during UNIT-OFF, via LOW-LEVEL int
;     Debounce is handled differently for EDGE-mode vs LEVEL.
;     For LEVEL debounce, we switch to IDLE mode until get a
      high-edge indicating (button release), then if no more
      edges are seen for 50mS, assume the button is debounced.
      If an EDGE-based request comes in, it MUST be to power
      off the device, so shift off LEDs, wait for button
      release, then wait 50mS; if not pressed then, power down.

OnOffButton:        ;external interrupt service routine
```

```
;-----------------------Utilities-------------------------------
--

GetLEDmode:
          ldi   temp0.0x01          ;address where we keep Mode
     #
          out   EEAR,temp0
          out   EECR,temp0
          out   EECR,temp0
          clr   temp0
          out   EEAR.temp0          ;wipe out address in case
of flaw
          in    LEDMODE,EEDR
          ret

SetUpLEDs:
          andi LEDMODE,MAXMODES-1   ;mask first!
          cpi   LEDMODE,0
          brne SUL1
          ldi   HiLights,Pattern0H
          ldi   LoLights,Pattern0L
          ret
SUL1:     cpi   LEDMODE,1
          brne SUL2
```


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```
                ldi  HiLights,Pattern1H
                ldi  LoLights,Pattern1L
                ret
        SUL2:   cpi  LEDMODE,2
                brne SUL3
                ldi  HiLights,Pattern2H
                ldi  LoLights,Pattern2L
                ret
        SUL3:   cpi  LEDMODE,3
                brne SUL4
                ldi  HiLights,Pattern3H
                Idi  LoLights,Pattern3L
                ret
        SUL4:   cpi  LEDMODE,4
                brne SUL5
                ldi  HiLights,Pattern4H
                ldi  LoLights,Pattern4L
                ret

        SUL5:   cpi  LEDMODE,5
                brne SUL6
                ldi  HiLights,Pattern5H
                ldi  LoLights,Pattern5L
                ret
```

```
SUL6:     cpi  LEDMODE,6
          brne SUL7
          ldi  HiLights.Pattern6H
          ldi  LoLights,Pattern6L
          ret
SUL7:     ldi  HiLights,Pattern7H
          ldi  LoLights,Pattern7L
          ret

     StartTimer:    :init TCNT0, TCCR0, TIFR, TTMSK.
          ldi  temp0.0x01      ;div-bv-1 = 0X01
          out  TCNT0,temp0     ;preload the count with a known
          val
          out  TCCRO.temp0     ;set to clk/1 mode
          ldi  temp0,0x02
          out  TIFR.temp0      ;force the flag off
          out  TIMSK,temp0     ;enable Timer int
          ldi  FlashCtr,(DutyFlash-1)   ;set up to turn on
LEDs soon
          ret

SaveMode: ;this saves the current LED mode to EE if necessary
          ldi  temp0.1
          out  EEAR.temp0      ;set address
```

```
                out   EECR,temp0      ;do read,
                out   EECR.temp0      ;twice
                in    temp0.EEDR      ;grab it
                cp    temp0, LEDMODE  ;same?
                breg  Smret           ;if so, exit
                out   EE DR, LEDMODE  ;put value
        ;
                ldi   temp0.4         ;compatibility for 8515
                out   EECR,temp0      ;if " " "

                ldi   temp0.2         ;(needed for both 8515 & 1200)
                out   EECR,temp0      ;force write
                nop
        SMwait: sbic  EECR.1          ;break if see LOW
                rimp  SMwait
        SMret:  ret
```

While this invention has been described in reference to illustrative embodiments, this description is not intended to be construed in a limiting sense. Various modifications and combinations of the illustrative embodiments, as well as other embodiments of the invention, will be apparent to persons skilled in the art upon reference to the description. It is

therefore intended that the appended claims encompass any such modifications or embodiments.